ORGANIC HEART DISEASES AND IMMUNITY FROM PULMONARY TUBERCULOSIS.[1]

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Whether organic heart diseases do or do not grant immunity from pulmonary tuberculosis depends upon the presence or absence of a coexisting long-standing passive hyperemia in the pulmonary circulation. This is the central thought of my topic, for it is just as demonstrable that the organic heart lesions accompanied by an anemia in the lungs, in the large majority of cases, are followed by pulmonary tuberculosis, as that those that produce a passive hyperemia either prevent its occurrence, or, if it has previously existed, retard its advancement or effect its extinction.

Before entering more in detail upon the topic of the paper, I desire briefly to call attention to a few well-known anatomical facts which intimately concern the subject in hand. The heart, having its right ventricle forcing the blood into the lungs, on the one side of the lesser circulation, and the left ventricle receiving its aërated blood on the other, because of this peculiar position of regulating the pulmonary blood supply, seems more intimately associated in a causal relation to certain diseases of the lungs (among them being tuberculosis) than any other organ. Again, the pulmonary circulation is peculiarly affected by the heart's action in that its arteries receive the direct full impact of each ventricular stroke, since there is no intervention of a long system of blood-vessels between them and the heart. Again, I would call attention to the fact that the pulmonary arteries, even to their finer branches, since they have walls much thinner than corresponding vessels in the systemic circulation, due to their poorly developed muscular coat, cannot accommodate themselves readily to variations in the blood-pressure, and hence are readily subject to passive stasis.

From the foregoing it is plain that even slight disturbances in the heart's action must exert a direct influence upon the pulmonary circulation. How readily then can it be understood that long-standing circulatory disturbances must leave their indelible stamp upon the pulmonary tissues.

Such disturbances arise chiefly from a deranged valvular mechanism of the heart (causing either stenosis or insufficiency or both) which eventually leads to one of two conditions in the lungs, an increased or a diminished blood supply.

A free entrance of blood into the pulmonary arteries with a retarded exit from the pulmonary veins through the left auricle, as occurs in a well-marked case of mitral stenosis, must lead to pulmonary stasis, a condition quite the reverse to that in which there is a hindrance to the passage of blood into the pulmonary arteries, as in a well-marked case of pulmonary stenosis.

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[1] Read before the Section on Medicine of the Academy of Medicine, New York City. March 20, 1906.

These two conditions thus are diametrically opposed to each other and statistics readily prove that the one just as surely grants immunity from tuberculosis as that the other predisposes to it. Therefore, in any discussion of whether organic heart diseases do or do not grant immunity from tuberculosis, diseases of the two sides of the heart must be kept distinctly separated. The question of immunity seems to resolve itself into the amount of blood within the pulmonary tissues; and if at this point I were to enter upon that fascinating study of the bactericidal properties of the blood, I fear I should be carried far beyond the scope and object of this short paper. For whether we adopt the theory of immunity as promulgated by Metchnikoff and based upon the phagocytic power of the microphages and macrophages, or Ehrlich's side-chain theory, based upon the chemical affinities of the blood, or better still the more probable one that combines both these theories, is at present immaterial.

The object of this paper is to show that those organic heart lesions accompanied by well-marked pulmonary stasis, thus concentrating in the lungs the immunizing agents of the blood, whatever they may be, are rarely followed by phthisis pulmonalis, or if this previously existed, they exert a salutary effect upon it, and that the reverse conditions frequently are followed by pulmonary tuberculosis.

Louis[1] was among the first to draw attention to the fact that stenosis of the conus arteriosus or the pulmonary orifice is occasionally followed by tuberculosis of the lungs. Stoelker,[2] in 1864, gathered from the literature 116 such cases, of which 16 (14 per cent.) had tuberculosis. Lebert,[3] in 1867, stated that fully one-third of such cases become complicated with tuberculosis. Bernard Schulze,[4] in 1891, gathered accurate statistics on the subject, and asserted that 83.3 per cent. of such cases are followed by tuberculosis of the lungs. Thus, as we approach the present time, when autopsies are accurately performed and data carefully recorded there seems little doubt that pulmonary stenosis, with its consequent pulmonary anemia, is followed, in the large majority of cases, by pulmonary tuberculosis. As is well known pulmonary stenosis is almost always congenital; as a condition secondary to pulmonary tuberculosis it has never been recorded and very likely does not occur.

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[1] Memoires ou recherches anatomico-pathologiques sur la Phthise, Paris, 1826.

[2] Ueber angebornere Stenose der Arteria Pulmonalis, Berlin, 1864.

[3] Einfluss der Stenose des Conus Arteriosus, u. s. w. auf Entstehung der Tuberculose, Berl. med. Woch., 1867.

[4] Beiträg zur Statistik der Tuberculose verbunden mit Herzklappenerkrankungen, Inaugural Dissertation, Kiel, 1891.

As regards the left side of the heart, Rokitansky[1] was the first to draw attention to the fact that disorders of the heart leading to pulmonary stasis are rarely associated with tuberculosis, saying: "Cyanosis or rather every disease of the heart, vessels, and lungs that causes cyanosis is incompatible with tubercle formation; that is, it offers an extraordinary immunity from tuberculosis." This statement has been the subject of much discussion and no little opposition, but in the main its truth holds as good today as when it was first promulgated. In general it has been supported by Traube,[2] Peter,[3] Fraentzel,[4] Kryger,[5] Eichhorst,[6] Otto,[7] Bamberget,[8] Eymann,[9] and others, although there have been minor differences of opinion. Frommolt,[10] in 1875, stated that the co-existence of chronic valvular disease of the heart and pulmonary tuberculosis is by no means as rare as was formerly supposed; but inasmuch as in many of his cases secondary changes, such as hypertrophy and dilatation of the heart (especially of the right ventricle), were not marked, there was little reason to expect the development of the circulatory changes in the lungs (cyanosis) that give rise to the immunity from tuberculosis. Kryger, apropos of cases in which healed pulmonary tuberculosis is associated with valvular disease of the heart, states his belief that most frequently the restraint and healing of the tuberculosis is dependent upon the heart disease.

There remains still to discuss the differences in the incidence of pulmonary tuberculosis in cases of aortic and of mitral valvular disease. Frommolt, somewhat at variance with Traube, states that "diseases of the aortic valve complicated with pulmonary tuberculosis occur somewhat more frequently than those of the mitral valve; still the difference is insignificant." His statistics are: Total number of autopsies, 7870; cases of mitral stenosis, 76; combined with tuberculosis of the lungs, 4 (5 per cent.); cases of mitral insufficiency, 76; combined with tuberculosis of the lungs, 8 (10 per cent.); total for all mitral lesions, 15 per cent. Cases of aortic stenosis, 33; combined with tuberculosis of the lungs, 2 (6 per cent.); cases of aortic insufficiency, 52; combined with tuberculosis of the lungs, 7 (13.5 per cent.); total for all aortic lesions, 19.5 per cent. Traube would lead one to believe that aortic insufficiency is not infrequently followed by tuberculosis of the lungs, since he states that he has seen a number of such cases. Traube's statement, based upon a large clinical experience, must receive due consideration. The discrepancy between his and Frommolt's statement may be accounted for in two ways:

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[1] Oesterreichischer Jahrbücher, 1836, vol. xvii.

[2] Gesammelte Beiträge zur Pathologie und Physiologie, 1871, vol. ii.

[3] Gazette des Hôpitaux, 1876.

[4] Krankheiten des Herzens, 1899, vol. ii.

[5] Ueber das gleichzeitige Vorkommen von Lungentuberculose und Klappenfehlern des linken Herzens, Inaugural Dissertation, München. 1889.

[6] Handbuch der speciellen Pathologie und Therapie, 1889, Band i.

[7] Das Ausschliessungsverhältniss zwischen Herzklappenfehlern und Lungenschwindsucht, Virchow's Archiv., vol. cxliv.

[8] Lehrbuch der Krankheiten des Herzens, 1857.

[9] Ueber die Combination von Phthise und Herzfehler, Inaugural Dissertation, Würzburg, 1886.

[10] Ueber das gleichzeitige Vorkommen von Herzklappenfehlern und Lungenschwindsucht, Archiv der Heilkunde, 1875, vol. xvi.

1. Frommolt's conclusion is based upon actual postmortem findings, whereas Traube's is largely upon clinical observation, and the fact must not be overlooked that even the best of diagnosticians have been misled in such cases as we are now considering, by accidental murmurs and falsely enlarged boundaries of the heart, owing to tuberculous consolidation of the on-lying borders of the lungs. Recently, I was called upon to decide the presence or absence of an organic heart lesion in a case of advanced phthisis pulmonalis. Auscultation showed a very loud, coarse, rasping, systolic murmur, localized at the apex. (Its timbre was that of an organic and not an accidental murmur.) The boundaries of the heart could not be outlined owing to consolidation of the on-lying borders of the lungs. The second pulmonic sound was accentuated. A diagnosis of organic heart lesion of the mitral valve had been made. I did not coincide with this diagnosis on the grounds: first, that the association of organic disease of the heart and advanced tuberculosis is very uncommon; second, that the heart boundaries could not be outlined; third, that the murmur remained localized, not being transmitted in any direction. Later, the autopsy proved a normal endocardium throughout and the mitral valve smooth and thin. There *was* advanced fatty degeneration of the heart muscle and a *relative mitral insufficiency* in consequence. I speak of this case as one of many in which the clinical diagnosis of organic heart disease of the endocardium in cases of advanced phthisis pulmonalis is not substantiated at the autopsy, and offer it as a warning against forming too hasty conclusions from the findings of the too oft misleading stethoscope.

2. Frommolt himself admits that in many of the cases from which he drew his conclusions, the "*sine qua non*," namely, well-marked secondary changes of hypertrophy and dilatation were absent. How decidedly necessary these are, at least in cases of mitral stenosis, Traube emphasizes.

However, Traube's statement that he never saw a case of tuberculosis following a mitral stenosis with well-marked hypertrophy of the right ventricle must remain an individual experience, for in this extreme none of the authorities seem to concur.

Bernhard Schulze's statistics are: Total number of autopsies, 6535; mitral lesions, 42; combined with tuberculosis, 2 (4.76 per cent.); aortic lesions, 52; combined with tuberculosis, 8 (15.4 per cent.).

In concluding the subject I offer the following summary:

1. Organic valvular diseases of the right side of the heart that produce pulmonary anemia afford conditions favorable for the ready growth of the tubercle bacillus and are as a fact most frequently associated with pulmonary tuberculosis.
2. Organic valvular diseases of the left side of the heart when accompanied by well-marked secondary changes of hypertrophy and dilatation, producing long-standing passive hyperemia of the lungs, are very rarely followed by tuberculosis of the lungs and not infrequently effect an extinction of a previously existing tuberculosis.
3. The exceptional cases that do arise must be accounted for by certain qualitative and quantitative changes in the bactericidal properties of the blood, reducing its immunizing powers.

And now, a practical point in the treatment of at least incipient phthisis suggests itself.

Nature clearly points the way in two instances: chronic emphysema and those valvular heart lesions that produce long-standing pulmonary congestion. In both these instances the autopsy frequently has revealed quite extensive tuberculous processes that have become entirely extinct. On the other hand, all pathologists and clinicians of much experience agree that tuberculosis rarely follows these two conditions.

What is the chief underlying pathological condition produced by these two affections? A constant passive congestion of the lungs.

What does this mean from the standpoint of immunity or therapeutics? That there must be collected in the lungs and kept there constantly an increase of those agents of the blood that are antagonistic to the growth of bacterial life.

When we have the reverse conditions, nature's struggles against the white man's scourge are indeed too sad to relate; frequently the autopsy reveals in persons in the prime of life afflicted with chlorosis or general anemia, primary or secondary, lungs that have become completely destroyed by tuberculcsis within a few weeks.

All this suggests in the treatment of our cases of at least incipient pulmonary tuberculosis physical exercise in the open air, several times a day, sufficient to produce severe congestion of the lungs; not mere gentle walking or driving, but running until the induced congestion of the lesser circulation interferes with the free action of the right ventricle and forces temporary rest. There never was a truer remark, made half in jest, than that of Elbert Hubbard's, in his little monthly pamphlet, *The Philistine*, "Tuberculosis is but a form of laziness."

Naturally, one must be governed in each individual case as to the character and amount of physical exercise prescribed. It is quite outside the scope of this paper to attempt to formulate rules of treatment or to exercise for others that judgment so requisite in the successful treatment of any disease.

I have been attempting to point out the conditions under which the autopsy discloses that nature frequently does effect a cure of phthisis pulmonalis even though this may have gained a very extensive foothold, as evidenced by Case I, herewith reported. To this frequent daily exercise must be added climatic conditions high in oxygen content, and therefore relatively free from germ life, together with plenty of such food as experience teaches will best build up and fortify nature's defences against microbial life—namely, the bactericidal properties of its blood.

I have intentionally spoken of incipient phthisis, being fully aware that the dangers of a sudden hemoptysis arising in the cases of advanced phthisis from the artificially produced local increased blood pressure caused by the severe physical exercise is a risk that few therapeutists would care to assume. Even in the case of incipient phthisis there is some risk that must be assumed in this respect, and it would be well to explain this to one's patient before beginning the treatment herein suggested.

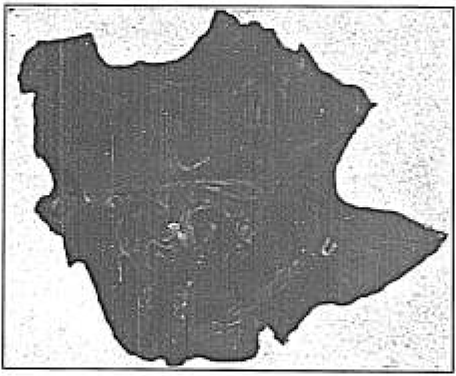
The following two cases illustrate typically the effect organic heart disease has upon the tuberculous processes of the lung, thus fittingly supporting the opinions expressed in this paper:

CASE I.—Metropolitan Hospital, New York City, November 11, 1905. M. B., aged forty-seven years, widow; occupation, general housework. Family and personal history are negative. Present illness began about six weeks ago, with a loose cough accompanied by sharp pains over anterior aspect of chest and a profuse whitish serous expectoration. No hemoptysis, no night sweats, no noticeable loss of weight. Subcutaneous and muscular tissues well-nourished. Extremities cold, cyanotic, and œdematous. Pupillary reflex is normal. Chest well-developed and symmetrical. Respiration regular, deep, and easy. Expansion good. Lower right lobe gave flat percussion note and auscultation revealed bronchial respiration. Harsh bronchial respiratory sounds over upper lobe of left lung. Sibilant rales in the right axilla.

Apex-beat not visible. Palpation showed it to occupy one finger's breadth in left midsternal line. At the apex were heard a presystolic and a systolic murmur transmitted to the left and a sharp accentuation of the second pulmonic sound. Pulse was regular, moderately full, and tense. Superficial arteries sclerosed. Sputum analysis on three different occasions showed absence of tubercle bacilli.

Autopsy, January 11, 1906. Postmortem rigor present. Hypostatic congestion in dependent parts of the body. Cyanosis of face, finger tips, and toes. Pupils each equally dilated about 4 mm. Slight œdema over both ankles and tibias. Petechial hemorrhages over anterior surfaces of both legs. Subcutaneous and muscular tissues well preserved. Abdomen prominent and fluctuates under palpation. Abdominal cavity contains fully 4000 c.c. of a clear light amber colored transudate in which appears no flocculent masses. Peritoneum is smooth, glistening, and transparent. Right pleural cavity contains about 500 c.c. of a light amber colored, partially turbid fluid, in which are floating flocculent recently formed fibrinous masses. Right pleura, over lower lobe of lung, has recently formed fibrinous masses attached to it and at the apex of the right lung are old tough pleuritic adhesions. Left pleural cavity contains very small amount of clear light-amber colored fluid containing no fibrinous flocculi. At the apex of the left lung are old tough fibrous pleuritic adhesions. Pericardium not adherent to either lung. Pericardial sac contains about 200 c.c. of a clear, light amber-colored transudate containing fibrinous flocculi. Pericardium (both layers) is smooth, glistening, and transparent. Veins of external surface of the heart are engorged. Coronary arteries show arteriosclerosis. Both ventricles are dilated. Heart weighs 285 grams. Firm in consistency. Heart muscle of rich, brown color. Left ventricular wall measures 1 cm. at its apex and 0.3 cm. at its base. Right ventricular wall measures 3 to 5 mm.; right ventricle is dilated. Left auricle is dilated. Endocardium throughout is smooth, glistening, and transparent. Tricuspid and pulmonary valves show no change. Aortic valve shows beginning atheromatous changes at its base and at the

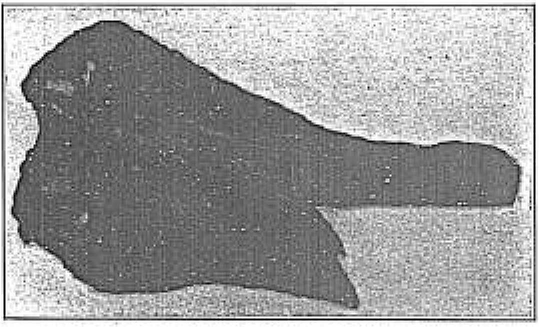
FIG. 1



Stenosis of the mitral orifice with newly formed vegetations upon an old thickened valve. The vegetations were provoked by pneumococci from a coexisting lobar pneumonia of the right lower lobe.

opening of the coronary arteries. Its closing margin is thin and free of growths. The valve is perfectly competent. The mitral valve considerably thickened along its closing margins, which is covered by recently formed vegetative growths and a few ulcerations (Fig. 1). The chordæ tendineæ are shortened and considerably thickened. The valve appears to be more stenotic than insufficient. Tissues of the pharynx and larynx show chronic congestion. Blood-vessels of œsophagus are dilated and engorged. Lungs: left upper lobe, at the apex, are several contractions from scar tissue and several extinct partially calcified tuberculous foci (Fig. 2), each well-defined from the surrounding normal tissue by a limiting wall of tough fibrous tissue. Balance of the lobe shows nothing abnormal. Lower lobe anteriorly and laterally near its upper margin also shows

FIG. 2

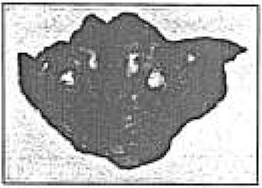


Apex of the left lung (Case I) showing healed tuberculous foci.

a few extinct tuberculous foci in every respect similar to those of the upper lobe. Balance of the lobe shows passive hyperemia and œdema. Nowhere in either lobe can any active tuberculosis be discovered. Right upper lobe at apices presents same appearances as corresponding left upper lobe (Fig. 3). One-half of the middle lobe is considerably contracted and contains tough, fibrinous connective tissue with numerous extinct calcified sharply circumscribed tuberculous foci (Fig. 4). The balance of this lobe is solid, finely granular, of dark-reddish color, evidencing red hepatization of acute lobar pneumonia. Lower lobe reveals the usual signs of red hepatization of acute lobar pneumonia. Nowhere throughout this lung can any active tuberculous process be discovered. The spleen, kidneys, and liver reveal the usual signs of cyanotic induration.

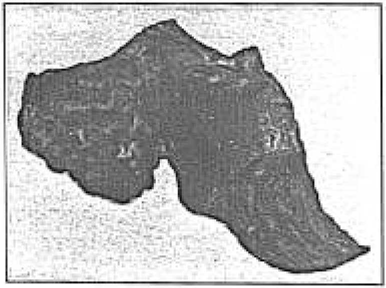
An acute splenic tumor is present. Stomach and intestines show chronic inflammation and passive congestion. Adrenals are normal. Microscopic examination of the portion of the lung affected by the pneumonia, as well as of the newly formed vegetative growths on the mitral valve, demonstrated a diplococcus similar to the Fraenkel-Weichselbaum diplococcus.

FIG. 3



Apex of the right lung (Case I) showing healed tuberculous foci.

FIG. 4



Middle lobe of the right lung (Case I) showing healed tuberculous foci and fibrosis. The dark portion in the centre is acute lobar pneumonia (which also involved the entire lower lobe of the right lung).

Diagnosis. Acute malignant ulcerative endocarditis of the mitral valve upon an old chronic inflammatory thickening; mitral stenosis with insufficiency; extinct tuberculosis of both lungs; acute lobar pneumonia of the right lower lobe and one-half of the right middle lobe; chronic cyanotic induration of liver, spleen, and kidneys; acute splenic tumor.

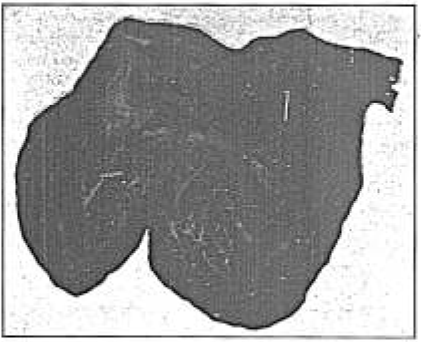
CASE II.—Metropolitan Hospital, New York City, December 18, 1905. W. S., aged fifty-one years, Ireland, salesman. Family history negative. In childhood had measles, scarlet fever, and whooping-cough. When twenty-one had gonorrhœa. Five years ago had acute inflammatory rheumatism. On October 20, 1905, as result of exposure had severe pains in chest, with cough, fever, and night sweats. Recovered from this attack and upon a second exposure suffered a relapse with same symptoms. Bowels regular, appetite poor, sleep broken. Œdema of both legs. All reflexes normal. Chest well-developed and symmetrical. Respiratory movements easy, regular, twenty-four per minute. Percussion and auscultation of chest negative, except a few crepitant rales at the bases of both lungs. Apex-beat: point of maximum impulse at sixth interspace left midsternal line. Right border of the heart at right sternal line. Left border reached 1 cm. to left of the midsternal line. A loud systolic blowing murmur heard at apex and transmitted to the left axilla. At the second right intercostal space close to the sternum could be heard a loud rasping systolic murmur transmitted upward into the carotids. The second pulmonic sound was loudly accentuated. Pulse regular, 90 per minute, hard and non-compressible. Lower border of the liver one finger's breadth below the margin of the ribs.

On February 11, 1906, patient had an attack of erysipelas of the face which increased in severity, bringing on delirium and causing death on February 13, 1906. Repeated examination of the sputum failed to detect any tubercle bacilli.

Autopsy. February 14, 1906. Body pale. Slight cyanosis of finger tips. Postmortem rigor present. Hypostatic congestion in the independent portions of the body. Pupils both equally dilated, 6 mm. Face deeply congested and cyanotic, especially about eyes and cheeks. Abdomen prominent and fluctuates on palpation. It contains about 2000 c.c. of a pale, light amber colored transudate. Peritoneum everywhere smooth, glistening, and transparent. Mesentery and mesentery glands normal. Diaphragm on right side at upper border of the fifth rib, and on the left side at the sixth rib. Each pleural cavity contains about 500 c.c. of a pale, light amber colored transudate. Pericardial sac about 30 c.c. of fluid. Pericardium is smooth, glistening, and transparent. Both ventricles of the heart are widely dilated. The veins on the external surface of the heart are engorged. Coronary arteries are slightly arteriosclerotic. The blood in both ventricles is fluid. Heart weighs 450 grams, soft in consistency, and of a light yellowish-brown color. The left ventricle widely dilated. Its wall measures at apex 1.5 cm. and at its base 2 cm. Right ventricle widely dilated. Its wall measures at apex 3 mm. and at its base 5 mm. The tricuspid and pulmonary valves are normal. The three cusps of the aortic valve are chronically thickened at their bases and their free margins are covered with numerous very large vegetative growths, some measuring 1 cm. in length. These growths very much narrow the opening of this valve (Fig. 5). The posterior wall of the aorta above the left cusp shows recently formed vegetative growths due to an aortitis. The mitral valve admits the tips of four fingers. Its edges are smooth and thin (relative insufficiency). The left auricle measures from 1 to 4 mm. in thickness. The right from 1 to 2 mm.

Lungs. The apex of the left lung shows well-circumscribed extinct calcified tuberculous foci and considerable fibrous connective tissue. Balance of this lung shows intense congestion and œdema. The right lung shows the same appearances as the left. The liver, spleen, and kidneys show chronic cyanotic induration. There is a very extensive anemic infarct of the spleen which measures 2.5 cm. in diameter on the surface of the spleen, and is 3 cm. in depth. There are old anemic infarcts in both kidneys. The stomach and intestines show chronic congestion. On the internal surface of the left half of the dura mater is a well-defined internal hemorrhagic pachymeningitis. The brain is œdematous, but otherwise apparently normal.

Fig. 5



Aortic stenosis with extensive vegetations along the free margins of the valve leaflets. The left ventricle being laid open, shows the extensive dilatation; there was also fatty degeneration of the myocardium. The apices of both lungs showed healed tuberculous foci.

Diagnosis. Aortic stenosis and relative mitral insufficiency; advanced fatty degeneration of heart muscle; passive congestion and œdema of both lungs with extinct tuberculous processes in both apices; cyanotic induration of liver, spleen, and kidneys; anemic infarcts of spleen and kidneys; pachymeningitis hæmorrhagica interna; œdema of the brain.